Semen Parameters and Hormone Profile of Men Investigated for Infertility at Midland Fertility Centre, Ilorin, Nigeria

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Abstract: This study aimed at comparing the semen parameters and pattern of endocrine abnormalities in patients investigated for male infertility in our fertility centre. Hormonal assays and semen analysis were carried out, from February 2008 to April 2010. Three hundred and sixteen (316) men were evaluated out of which forty-five (45) underwent hormonal assessment, because of the abnormalities in their sperm count. Sixteen (35.6%; 16 out of 45) of the subjects were azoospermic; twenty-three (23 out of 45; 51.1%) were oligospermic with sperm count less than 20 million sperm cell/ ml; and six (n=6; 13.3%) of the subjects were normospermic, with sperm count greater than 20 million sperm cell/ ml. The mean values of the hormonal assays for azoospermia were: 21.84±1.94 (luteinizing hormone), 14.14±4.4 (follicle-stimulating hormone), 23.95±17.43 (testosterone), and 15.03±2.91 for prolactin. Hormonal assays for patients having oligospermia were as follow: luteinizing hormone (LH) 12.56±3.90, follicle-stimulating hormone (FSH) 5.96±1.17, testosterone 10.13±2.40 and prolactin 13.42±2.43, while normospermic patients had the following hormone levels: LH: 7.72±3.90, FSH: 2.93±.74, testosterone: 17.00±3.36 and, prolactin: 12.45±2.16. We conclude that infertile men with low sperm count may not always present with abnormal hormone profiles; and, male factor, in this environment, contributes a high percentage to infertility.

Keywords: Azoospermia, Hormone profile, Normospermia, Oligospermia.

INTRODUCTION

Infertility is defined as the inability of a couple to conceive after at least 12 months of unprotected sexual intercourse. It occurs worldwide but differs in incidence and prevalence. Infertility is a common gynaecological problem affecting 15% of couples attempting their first pregnancy, in which case it is called primary infertility; while those with secondary infertility are about 10% of the population. Secondary infertility could be as high as 52% in some sub-Sahara African countries and as low as 23% in some Asian countries [1].

The perception of people about infertility differs from culture to culture. In the African setting where high premium is placed on procreation [2], infertility is an object of social stigma [3]. The attendant emotional, psychological, cultural and social burdens drain the couple of self belief and esteem [2].

Two principal factors are taken into consideration with regards to aetiology: male causative factors and female causative factors. Male infertility is associated with a reduction in the quality of sperm. A male causative factor is associated with 50% of all infertility cases [4], such that about 30% of the cases of infertility are associated with male causative factors, while 20% are associated with combined male and female factors [4]. A study amongst north-eastern Nigerians found combined male and female factors in 30% and male causative factors in 28.6% [5].

Semen analysis and hormone evaluation are essential parameters in giving a definitive diagnosis in infertile males [6]. Sperm characteristics include volume, pH, sperm concentration, motility, progressivity, morphology, and vitality. Azoospermia refers to absence of spermatozoa in the semen ejaculate, while in oligospermia, the count is less than 20 million/ml.

Abnormal hormone production has been noted as a male causative factor [7], and hormonal replacement could play a corrective role [8]. The most essential hormones to be evaluated include, follicle-stimulating hormone (FSH), luteinising hormone (LH), testosterone and prolactin; others are estradiol and thyroid hormone [9]. Decrease in sperm count is associated with low testosterone level [10].

Changes in FSH and LH could result in abnormalities of spermatogenesis in patients with low sperm counts [11], and very high levels of serum prolactin has been associated with infertility, hypogonadism, impotence, and galactorrhea [12].

Hypothalamus controls aspects of reproduction, including gametogenesis, cyclic variations and the development and maintenance of secondary sexual features. Hypothalamic stimulation may induce

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Table 1: Seminalysis and Hormone Profiles of the Studied Subjects (mean±SEM; P<0.05)

Remarks	Frequency (n)	Count x10^6	Motility in x10^6	LH	FSH	Test	PRL
Normospermia	6	44.17 ±7.82	29.20 ±4.45	7.72 ±3.09	2.93 ±0.74	17.00 ±3.36	12.45 ±4.89
Oligospermia	23	8.40 ±2.19	3.62 ±1.13	12.56 ±3.90	5.96 ±1.17	10.13 ±2.40	13.42 ±2.43
Azoospermia	16	0.00	0.00	21.84 ±12.5	14.14 ±4.4	6.63 ±2.09	15.03 ±2.91

receptivity in females and simple copulatory movements in males. Some hypothalamic neurons are sensitive to circulating oestrogen or testosterone [13].

Spermatogenesis is regulated by luteinizing hormone (LH) produced by the pituitary. LH binds to receptors on Leydig cells and stimulates testosterone production, which in turn binds to Sertoli cells to promote spermatogenesis. Follicle stimulating hormone (FSH**)** is also essential because its binding to Sertoli cells stimulates testicular fluid production and synthesis of intracellular androgen receptor proteins [14].

Testosterone regulates its own secretion by negative feedback mechanism. It acts on hypothalamus and inhibits the secretion of luteinizing hormone-releasing hormone (LHRH). When LHRH secretion is inhibited, LH is not released from anterior pituitary, resulting in the stoppage of testosterone secretion from testes. On the other hand, when testosterone production is low, lack of inhibition of hypothalamus leads to secretion of testosterone through LHRH and LH [13].

MATERIALS AND METHODS

This study was conducted at Midland Fertility Centre, Ilorin, Nigeria, from February 2008 to April 2010. A total of 316 patients were investigated within this period. However, this report was based on forty-five (45) subjects on whom both sperm analysis and hormone assays were conducted, as part of their investigations for infertility. The World Health Organization method [15] was adopted for semen analysis. For each sample, the colour, viscosity and liquefaction time were recorded. The volumes were measured using a graduated glass pipette. The sperm concentration was counted in million/ml using Mackler counting Chamber (Semen Analysis Chamber, ISO 9001:2000). The Olympus® Binocular microscope with magnification (x100) was used to observe the sperm cells. The motility, morphology and progressivity were also observed.

Hormone profile was carried out using a non-competitive (sandwich) ELISA kit, and read with the aid of a microplate reader (Model RT-2100C). The hormones analysed include: follicle stimulating hormone (FSH), luteinising hormone (LH), testosterone and prolactin.

The data were presented as mean±SEM, and analysed statistically by the application of student's t-test as described by [16].

RESULTS AND DISCUSSION

A total of 316 men were investigated for infertility during this study but 45 men had their hormonal status assessed. Out of the 45 men assessed, 16 (35.6%) were azoospermic, 23 (51.1%) were oligospermic, and 6 (13.3%) were normospermic; their respective sperm count and motility are shown in Tables **1** and **2**.

Table 2: Number of Male Partner within the Remarks of Seminal Profiles

	Frequency	Percentage (%)
Azoospermia	*16*	*35.6*
Oligospermia	*23*	*51.1*
Normospermia	*6*	*13.3*
Total	*45*	*100.0*

For the normospermic patients, the hormone status is as follows: LH: 7.72±3.09; FSH: 2.93±0.74; Testosterone: 17.00±3.36; Prolactin: 12.45±4.89; for oligospermic patients the hormone status is as follows: LH: 12.56±3.90; FSH: 5.96±1.17; Testosterone:

Table 3: Number and Type of Endocrinopathy

Hormones	Frequency	Percentage (%) Normal	Percentage (%) Abnormal
LH	24N, 21A	53.3	46.7
FSH	39N, 6A	86.7	13.3
Testosteron e	16N, 29A	35.6	64.4
Prolactin	18N, 27A	40.0	60.0

N-Normal; A-Abnormal

Table 4: Hormone Status Classified as 'Normal or Abnormal'

Status	Number	Percentage (%)
Normal Hormonal Status	24	53.3
Abnormal Hormonal Status	21	46.7
Total	45	100.0

10.13±2.40; and Prolactin: 13.42±2.43, while the hormone profile for patients having azoospermia showed LH: 21.84±12.5; FSH: 14.14±4.4; Testosterone: 6.63±2.09; and Prolactin: 15.03±2.91 (Table **1**). Only twenty-four (53.3%) out of the 45 patients had some degrees of derangement in their hormonal status, while the remaining 21(46.7%) were normal (Table **4**). Most of the patients (86.7%) had normal levels of FSH, while only 53.3% had normal levels of LH. Testosterone and prolactin levels were abnormal in 64.4% and 60% of patients respectively (Table **3**).

Majority of the males investigated in this study had oligospermia (51.1%), and more than one-third (35.6%) had azoospermia (Table **2**), making a total of 86.7% patients with sperm abnormalities (oligospermia and azoospermia). An earlier study by [17] observed a lower percentage (58.8%) of severe oligospermia and azoospermia in patients with abnormal hormonal pattern while 41.1% of the cases of severe oligospermia and azoospermia had normal hormone pattern.

The prevalence of endocrinopathy in men in the present study was lower than that reported in Kenya [18], in East Africa. Geidam *et al* (2008) in their study observed higher percentage values for Prolactin (53.1%) [19], LH (54.2%), and Testosterone (43.7%), but very low value of normal FSH (29.2%), compared with the current investigation (Table **3**). The normal hormone pattern seen in some of the patients may be related to either obstructive azoospermia or retrograde ejaculation and further evaluation in form of vasography, testicular biopsy and post-coital urine wash may be needed.

Male infertility is a common problem with a complex aetiology, requiring a complex andrological work-up in some cases for proper diagnosis [19]. This study showed the importance of proper endocrinological work-up in the evaluation of patient with male infertility when appropriate because these cases may have recognizable endocrinopathy that may be correctable.

Patients presenting with infertility sometimes have normal sperm characteristics. Studies by Oghagbon *et al* [20] showed that 27.6% of cases investigated presented with normospermia, while studies by Fadahunsi *et al* [21] and Abdulhadi *et al* [22] revealed 43.7% and 25% cases of normospermia respectively. However, studies from our Fertility Centre shows that a lesser percentage (13.3%) of patients investigated for male infertility had normal sperm characteristics. Oligospermia was found to be more in the current study (51.1%) compared with findings by Oghagbon *et al* and Fadahunsi *et al* [20, 21], who recorded 49% and 42% respectively. Studies by Abdulhadi *et al* in Kano-Nigeria showed a lower value of oligospermia (33.3%) [22]. The current study revealed that 35.7% were having azoospermia closely related to the study conducted in South-West Nigeria where 35% of patients had azoospersmia [23], but the studies conducted by Fadahunsi *et al* [21] revealed a lower percentage of 13.9%. The prevalence of high percentages of azoospermia in this study and similar studies suggests an increasibg rate of azoospermia. The two major causes of azoospermia are failure of spermatogenesis and bilateral ductal obstruction [20]. Azoospermia in Nigeria is due to failure of spermatogenesis [24]. According to Ojengbede *et al.*, azoospermic patients have prior sexually transmitted diseases which have been linked to seminiferous tubular damage and infertility [25, 26].

Men aged forty-two (42) and above were azoospermia. Perhaps, our men show up late to the hospital for investigation or marrying late. It has been shown that semen qualities deteriorate by as much as 3% per year [27]. This factor, in addition, probably contributes to the high percentage of abnormal spermiogram among our subjects.

Conclusively, for proper investigation of infertile couples, men must be thoroughly investigated because their contribution to infertility is high, and they must present themselves early enough for proper investigation.

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